SUMMARY - USU Air Quality Symposium, September 21-22, 2005

Cache Valley was the beneficiary of the visits of four nationally renowned air-quality experts. Kent Pinkerton, Frank Mitloehner, and Tony Wexler are from UC Davis and the San Joaquin Valley of California. C. Arden Pope is from BYU.

Kent Pinkerton does toxicology research on the effects of air pollutants on lung tissues. He presented the results of some of his studies in rats and primates and discussed the health effects of PM in children and adults. Frank Mitloehner studies dairy emissions and has an in-depth understanding of ammonia production from bovine waste products. He discussed sources of ammonia emissions and strategies that could be used to minimize ammonia emissions from waste products of cows. Tony Wexler has an in-depth understanding of particulate chemistry and discussed secondary formation of ammonium nitrate particulate and factors that promote its formation and affect its deposition and dissolution. C. Arden Pope is an economist that has international acclaim for his experience studying large populations of humans and the effects of air pollution on health.

Each of these scientists brought a unique set of experiences and understanding concerning particulate air pollution. Their areas of expertise were diverse but complementary in helping community leaders, Utah State students, faculty, and Cache Valley citizens understand the serious nature of particulate pollution as well as effective strategies for mitigation of the problem.

Below is a summary of significant data and salient points presented by each of these experts.

C. Arden Pope, Professor of Economics, BYU

-Wasatch Front studies consistently show that children exposed to PM10 on an acute basis have significant increases in respiratory symptoms (cough, asthma) and significant reductions in objective pulmonary function measurements (PEF).
-Studies done on the acute effects of PM exposure show that there are significant acute increases in cardiovascular and respiratory mortality when PM levels are elevated.

Cause of Death	**% of total deaths**	**Cause-specific percent increase per 50 mg/m3 increase in PM2.5**	**% of excess deaths due to PM exposure**
All cause	100	7.0	100
Respiratory	8	25.0	28
Cardiovascular	45	11.0	69
Other	47	0.4	3

Source: Pope. Environmental Perspectives 108(suppl4):713-723(2000)

-Long term "lower level" exposure also results in increased long-term mortality
-16 year prospective cohort study involving one-half of a million people in 156 metropolitan areas showed a 14% increase in death from lung cancer and a 9% increase in cardiopulmonary mortality for each 10ug/m3 increase in average levels of PM10.

Table 2. Adjusted Mortality Relative Risk (RR) Associated With a 10-μg/m³ Change in Fine Particles Measuring Less Than 2.5 μm in Diameter

	Adjusted RR (95% CI)*		
Cause of Mortality	1979-1983	1999-2000	Average
All-cause	1.04 (1.01-1.08)	1.06 (1.02-1.10)	1.06 (1.02-1.11)
Cardiopulmonary	1.06 (1.02-1.10)	1.08 (1.02-1.14)	1.09 (1.03-1.16)
Lung cancer	1.08 (1.01-1.16)	1.13 (1.04-1.22)	1.14 (1.04-1.23)
All other cause	1.01 (0.97-1.05)	1.01 (0.97-1.06)	1.01 (0.95-1.06)

*Estimated and adjusted based on the baseline random-effects Cox proportional hazards model, controlling for age, sex, race, smoking, education, marital status, body mass, alcohol consumption, occupational exposure, and diet. CI indicates confidence interval.

Source: Pope et al. JAMA March 6, 2002 – Vol 287 (9)

-Accelerated atherosclerosis due to PM and other adverse effects are believed to be due to and underlying local and systemic inflammatory response to PM

- **CONCLUSION 1.** There is now substantial epidemiological evidence that PM is an environmental risk factor for cardiovascular mortality, pulmonary illness, and in children, reduced lung function and lung growth/development.
-**CONCLUSION 2.** Studies that explored pathophysiological pathways that link PM exposure and cardiopulmonary mortality risk suggest that there likely are multiple mechanistic pathways with complex interdependencies including:

- Accelerated progression of COPD,
- Modulated host defenses and immunity,
- Pulmonary Inflammation/Hypoxia
- Altered cardiac autonomic function,
- Pulmonary/Systemic Oxidative Stress/Inflammation
- Initiation and acceleration of atherosclerosis.
- Contribution to acute thrombotic complications

ADDITIONAL OBSERVATIONS AND ADVICE FROM DR. POPE:
- Average PM levels in Cache Valley may be a bigger cause for concern than the limited number of spikes in PM that occur during wintertime inversions.
- There is no "safe threshold" level for PM.
-Cache Valley's current average PM levels are not bad but they are not good either.
-When considering long-term adverse health effects (heart attacks, strokes, various lung disorders and death), living in an air shed with an average PM2.5 level of 20 ug/m3 is equivalent to living with a spouse that smokes 1 pack of cigarettes per day.
-Cache Valley's 3-year average PM level is currently 13 ug/m3. From a health effects standpoint, this level of PM exposure is comparable to living with a spouse that smokes ½ pack of cigarettes per day.

- Epidemiologic studies in areas with predominantly NH4NO3 PM show significant acute and long-term health effects similar to areas where PM is not predominantly NH4NO3. In other words, Cache Valley's PM is not less dangerous than PM in other areas.
- Do not overstate the danger of PM2.5 but do not understate it either.
- Cache Valley could decrease its long-term cardiovascular risk by 4-5% if it could decrease its PM2.5 average from13 mcg/m3 down to 8 mcg/m3.
-For more information refer to six cities study in NEJM 1993 ;329:1753-1759, and also JAMA 2002 Vol. 287, 1132-1141
- As a community, Cache Valley should look at the long-term picture and try to get a reduction in average PM levels.
-Avoid isolated focus on the short-lived spikes during wintertime.

Kent Pinkerton, Anatomy and Physiology: Veterinary Medicine, UC Davis (Corrected Version-OK for the public)

-Ammonium Nitrate predominate PM is a common problem in the western United States.
-Ammonium Nitrate-elemental carbon particulate causes cellular damage in otherwise healthy rats and primates, particularly at airway bifurcations, and in the proximal alveolar air exchange regions. Cellular injury caused by ammonium nitrate-elemental carbon is comparable to the injury caused by ozone exposure.
-elemental carbon by itself does not seem to cause airway injury
-Organic carbon is more irritating than elemental carbon
-Rats exposed to concentrated ambient PM from Fresno (predominantly ultra-fine ammonium nitrate particulate) resulted in significant damage and injury to pulmonary epithelial cells and alveolar macrophaages. Larger size particulate PM10 caused more of an inflammatory response and not as much cellular damage as the ultra-fine particulate

Additional Observations by Kent Pinkerton

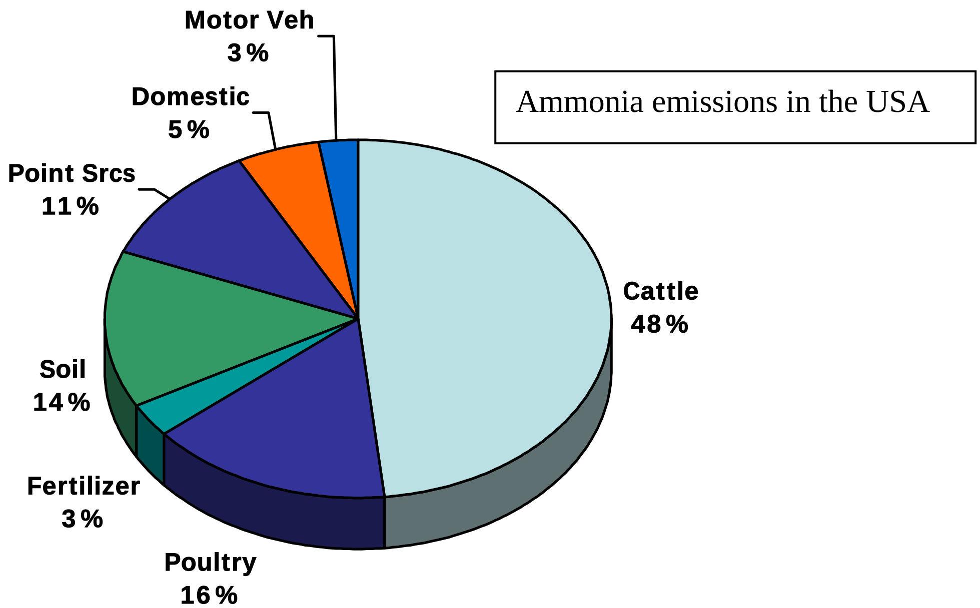
-The "Hygiene Hypothesis" does not likely apply to particulate exposure as children exposed to high levels of particulate tend to have more trouble with asthma when compared with children that are not exposed.
-**Small children** get as much as a **60-fold increase** in PM dose/kg body weight compared to adults breathing the same air
-80% of alveoli growth and development occurs after birth and PM exposure causes a demonstrable retardation in alveolar growth which results in measurable decreased lung capacity in children that live with elevated levels of PM2.5

Frank Mitloehner, Dept. of Animal Science, UC Davis (Corrected Version – OK for the Public)

-The average bovine produces 170 kg of nitrogen waste per year. Fifty percent of this nitrogen waste is in the form of urea in the urine. Seventy percent of the urea is converted to NH_3 by the action of urease enzymes in the feces.

-Ammonia production from bovine waste is at least 10-fold greater than human waste on a per capita basis (assumes usual mix of lactating and non-lactating animals)

-Cow waste products are responsible for 48% of ammonia emissions in the USA



-Ammonia is not released directly from a cow. The urea in the urine must be mixed with feces and hydrolyzed by urease in the feces before ammonia can be released.

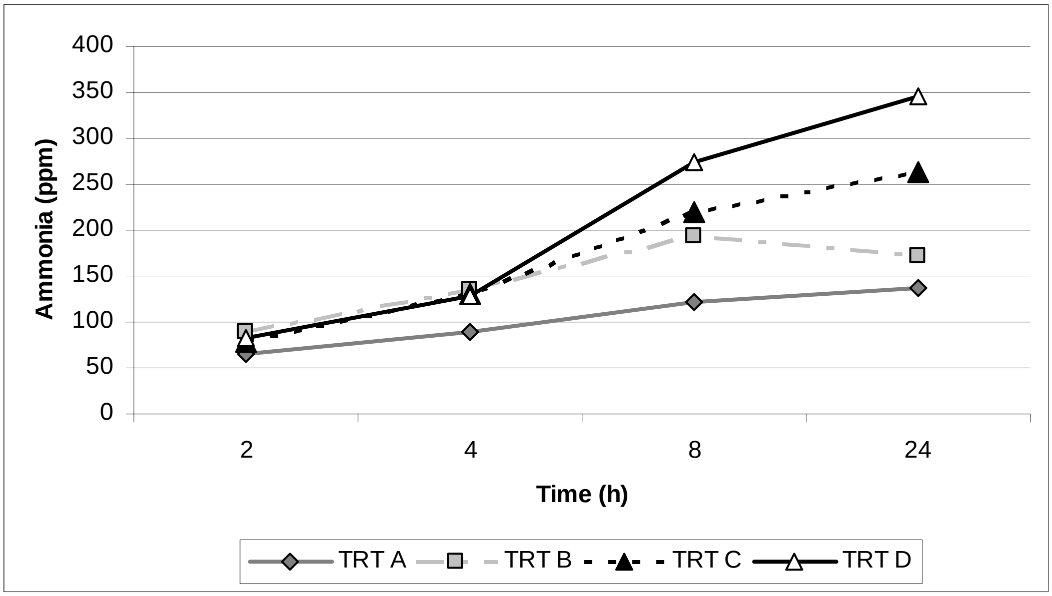
-This reaction occurs soon after feces and urine mix. Most of the urea gets converted to ammonia within 24-48 hours.

-The conversion of urea to ammonia is slowed by acidification of waste ($NaHSO_4$), lower temperatures, and separation of urine from feces.

-Ammonia formation in dairy lagoons is limited (2-15% of dairy ammonia comes from lagoons)

-Oxygenation of lagoons changes metabolism from anaerobic to aerobic and results in conversion of ammonia to nitrites and nitrates, limiting release of ammonia into atmosphere.

- Most cows are fed too much nitrogen. A 50% reduction in NH_3 can be achieved by reducing crude protein in feed from 18% to 16%.



Treatment Group A = Cows fed 14% crude protein
Treatment Group B = Cows fed 16% crude protein
Treatment Group C = Cows fed 18% crude protein
Treatment Group D = Cows fed 20% crude protein
There was no significant change in performance of cows fed 14% crude protein compared with those cows that were fed 20% crude protein.

-Most of ammonia emissions occur on the floor and flush lanes of free stalls and in corrals/feedlots. A relatively small amount of ammonia released from dairy lagoons.
-Shallow dairy lagoons with high pH release more ammonia per head than deep lagoons with low pH.
-Oxygenation of lagoons result in aerobic conditions with resultant fixation of nitrogen in the form of nitrates and nitrites, further reducing release of ammonia into the atmosphere.
- Land application of manure can result in nitrate and nitrite leaching in the ground water.
- VOC's from the rumen of a cow are mostly CH_4, acetone, methanol, ethanol, and acetic acid. These VOC's have a low to very low reactivity and are not a significant contributor to the formation of ozone or other reactive oxygen species.
- Elevated levels of NH_3 observed during Cache Valley winters are due mostly to the very small size of the air shed during inversions.
-In the long run, Cache Valley would likely benefit from reductions in both NH_3 as well as NO_x/nitrates. In the short term, NO_x reduction should be addressed first as it appears that at the present time there is excess ammonia and that nitric acid is the limiting factor in formation of particulate.

Tony Wexler, Mechanical and Aeronautical Engineering, UC Davis (correcte4d Version OK for the Public)

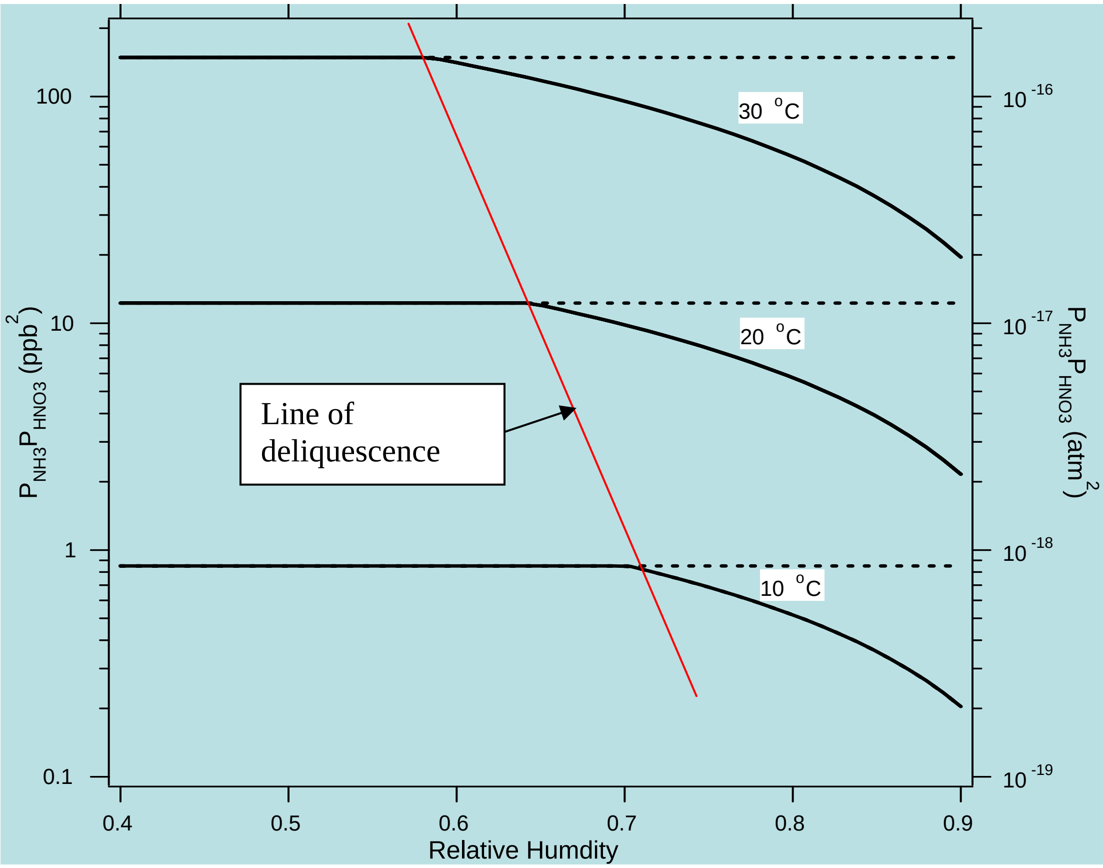
-Cache Valley's air quality problems are not unique. The air temperatures may be extreme but the chemistry, inversion problems, topography and makeup of wintertime PM is similar to that of other places such as California's Central Valley.

-60,000 people die each year in the USA due to air pollution particles alone.

- Formation of ammonium nitrate from NH3 and HNO3 happens on the surface of a pre-existing particle (elemental carbon, organic carbon, crustal elements). There is no significant gaseous phase NH4NO3.
- NH4NO3 in particles can exist as a solid when relative humidity (RH) is low, or in the aqueous phase when relative humidity is higher.
- Ammonium nitrate forms from NH3 and HNO3 on the surface of pre-existing particles (EC, VOC, dust) when the following conditions exist:

$$[NH3_{Total}][HNO3_{Total}] > K(_{\text{temp, relative humidity}})$$

- K is not a constant. K is a function of temperature and relative humidity. The value of K decreases with a drop in temperature or a rise in humidity. Hence lower temperatures and higher relative humidities promote formation of ammonium nitrate on the particle surface.



(Deliquescence is the conversion of solid phase to the aqueous phase as relative humidity increases.)

- Cache Valley's wintertime atmosphere is usually nitric acid limited due to relatively high levels (on a molar basis) of airborne ammonia.
- Nitric acid forms secondarily in the atmosphere from the following reactions:

Nighttime: $NO_2 + O_3 \rightarrow NO_3 + O_2$
$NO_3 + NO_2 \rightarrow N_2O_5$
$N_2O_5 + H_2O \rightarrow 2HNO_3$

Daytime: NO_x + Reactive VOC's + sunlight $\rightarrow O_3 + OH + HO_2$ (radicals)
$NO + O_3 \rightarrow NO_2 + O_2$
VOC + NO_x + sunlight $\rightarrow$ OH
$NO_2 + OH \rightarrow$

- VOC's are involved in the formation of HNO_3 from NO_x as can be seen in the above reactions.
-Wintertime reactive VOC's in Cache Valley are due mostly to the use of the internal combustion engine as VOC emissions from plants and other natural sources would be relatively low when the temperature is low.
-VOC's are diverse in their levels of reactivity and ability to generate radical chemical species in the presence of sunlight.
-Surface deposition of particles depends on their size.

- Turbulent flow transports all particles to near a surface
- Laminar sub-layer of air just above the surface is resistant to particle penetration
- Particles > 1 µm have enough kinetic energy to penetrate the laminar sub-layer
- Particles < 0.1 µm penetrate the sub-layer by Brownian motion/diffusion
- Particles between 0.1 µm and 1 µm do not penetrate the laminar sub-layer easily and are not readily deposited on a surface.
- Particles > 10 µm deposit on surface by gravitational settling.

-Airway deposition is similar to surface deposition and results in adverse health effects

- Settling and impaction for particles > 1 µm
- Brownian Diffusion for particles < 0.1 µm
- Low Deposition for particles in between these sizes

- "Low Hanging Fruit" in Cache Valley include:

Emissions Inspections to repair or remove the bottom 10-20% of vehicles that produce 45-65% of the NO_x and the VOC's
Reduced protein diet – feed the cows only what they need to be productive (14%)
Community efforts to reduce VMT's during inversions

Reference:
http:mae.ucdavis.edu/wexler/aim

Randy Martin, Civil and Environmental Engineering, USU
-Particulate levels during wintertime inversion conditions are generally high.
-Particulate levels are statistically uniform throughout Cache Valley during inversions
-Cache Valley will likely violate the NAAQS 24-hour standard during the winter of 2005-2006.
-Ammonia levels are very high in Cache Valley and significantly exceed levels of nitric acid (from NOx) on a molar basis. The formation of ammonium nitrate particulate is therefore nitric acid limited. Strategies to lower ammonium nitrate particulate must take this into account.
-The "dirtiest" 10% of cars in Cache Valley produce 45% of the total NOx and VOC emissions
-Indoor air has significantly lower levels of particulate when compared with outdoor air during wintertime inversions.

Phil Silva, Chemistry, USU
-Wintertime PM2.5 in Cache Valley is made up mostly of ammonium nitrate.
-The size of the particulate during wintertime inversions is relatively uniform and generally very small, between 0.1 and 1 micron in diameter.

In summary, community leaders, citizens, USU faculty and students learned the following:

1. Particulate air pollution levels like those seen in Cache Valley during wintertime inversions are a significant health hazard. Adverse health effects of acute high-level particulate exposure include acute respiratory symptoms, asthma attacks, breathing difficulties, variability in autonomic function, pollution event-associated increased risk of pneumonia, myocardial infarction, stroke, and death.

2. The health effects of long-term lower level particulate exposures are also significant. Average particulate (PM2.5) levels seen in Cache Valley are not low and the health effects of year-round living in Cache Valley are comparable to those associated with chronic daily exposure to ½ pack per day of second-hand cigarette smoke. These effects include reduced lung function and lung development in children, and increased risk of heart attack, stroke, lung cancer, and death.

3. Cache Valley's particulate problem is due mostly to secondary formation of ammonium nitrate. The ammonia is due mostly to hydrolysis of urea from cow urine. Nitric acid (nitrate) is formed by chemical reactions in the atmosphere involving nitrogen oxides (NOx) from car and truck exhaust, and ozone along with other reactive oxygen species (radicals). Reactive oxygen species are promoted by the reaction of sunlight with volatile organic carbon molecules (VOCs). VOCs and nitrogen oxides (NOx) come from operation of internal combustion engines.

4. Older and poorly maintained automobiles produce more VOC's and NOx than newer well-maintained vehicles. The bottom 10% of Cache Valley's fleet produces 45% of

NOx and VOCs. The bottom 20% of Cache Valley's fleet produces 65% of the NOx VOCs. (Data from 2004 on-road emission monitoring done by Dr. Randal Martin, USU)

5. Strategies to achieve sustainable reductions in NOx, VOC, and ammonia emissions should be developed and implemented in Cache Valley to reduce the short-term and long-term health effects caused by elevated levels of PM2.5.

6. "Low hanging fruit" in Cache Valley at the present time include:

-Reduction of vehicle emissions by implementation of an appropriate emissions inspection and vehicle maintenance program,

-Continued efforts at reduction of vehicle miles traveled (VMT) especially during wintertime inversions,

-Further investigation and education of the agricultural community with the goal of a reduction in ammonia emissions.

Reducing ammonia emissions may not result in a dramatic reduction in particulate, but it is a "cost-negative" strategy that would be financially beneficial to the agricultural community. It is also healthier for the cows and certainly would not make air quality worse.

7. Children and at-risk adults should continue to avoid unnecessary outdoor air exposure when levels of PM2.5 are elevated. Indoor air is likely to be significantly less dangerous than outdoor air during inversion conditions when particulate levels are elevated.

This summary was compiled by Edward Redd, MD, Medical Officer and Deputy Director of the Bear River Health Department. The content of this summary was reviewed, corrected, and approved for public use by C. Arden Pope, Kent Pinkerton, Frank Mitloehner, and Tony Wexler. If there is any question or concern regarding this document, please contact:

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1. American Heart Association Scientific Statement: Air Pollution and Cardiovascular Disease. Brook et al. *Circulation* June 1, 2004;109:2655-2671. Also available in a PDF format for free on the internet at http://circ.ahajournals.org/cgi/reprint/109/21/2655